matters, in the stomach or the contents of the stomach, or in the intestines or in their contents, can no longer be considered as any judicial scientific proof that the poison was introduced into the system by the alimentary canal at any part, as has been assumed. (Absolute.) 5. That antimony, being absorbed with great rapidity wherever introduced, the point of surface at which it is taken into the system may afford slighter indication of the presence of the poison than any other parts of the organism: *ergo*, that the point of introduction can never be proved by mere chemical analysis. (Absolute.) 6. That antimony applied locally, so as to admit of being rapidly absorbed, seems to excite but little amount of local injury, although it exerts marked local effects when brought by the blood to any surface for elimination: *ergo*, that the appearance of intense redness or inflammation in the stomach or other part of the alimentary canal, in supposed cases of death from antimony, is no scientific proof, nor yet indirect evidence, that the poison was received into the system by this canal. (Absolute.) 7. That the symptoms of poisoning by antimony by large doses are, as a general rule, those of vomiting, purging, and rapid collapse; and that the same symptoms, somewhat modified in their course, result from small doses repeated frequently during a prolonged period. 8. That to this rule exceptions occur: to wit, that antimony, when thrown into the system in a large dose, and in such a way as to prevent its digestion, as by direct injection into the veins, may destroy the muscular power so suddenly that the symptoms of vomiting and purging may not present themselves. And, again, that when introduced very slowly, as by application to a small wound, it may also destroy by producing simple exhaustion, without the specific symptoms of purgation or vomiting. 9. That, in all forms of antimonial poisoning, death occurs mainly from failure of the circulation; the respirations being continued after the cessation of the heart's beat. 10. That the pathological appearances incident to antimonial poisoning are—(*a*) general congestion; (*b*) marked fluidity of the blood; (*c*) intense vascularity of the stomach in the course of the greater curvature, and, in some cases, of the rectum and other parts of the canal, but without ulceration; (*d*) a peculiarly pale yellow or occasional dark glairy secretion on the alimentary surface. Lastly, contrary to the statements of Magendie, antimony seems to excite no other pulmonary lesion than simple congestion. 11. That the election of antimony by different parts of the body is as yet an open question; that the liver, however, would appear to be the structure in which it is most collected when the administration is slow and in small doses; and that the elimination of the poison is attempted by all the secreting surfaces. 12. That, in rapid poisoning, the fatal effect seems due to direct chemical changes in the blood, and to indirect effect therefrom on the heart; while, in slow poisoning, there is superadded an interference with the assimilative powers, the result of the lesions excited in the stomach and other parts of the alimentary canal.

We have further to remark that, in animals dosed for a few days with antimony, and then kept for periods of seven, fourteen, and twenty-one days, antimony was found in each case in abundant proportions in the liver, and in smaller proportions in the kidney and heart; and also in the contents of the stomach in cases where the animals were destroyed during digestion of food.

The "tolerance" of antimony seems to us to depend entirely on the free elimination of the poison by the kidney.—*B. and F. Med. Chirurg. Rev.*, Oct., 1856.

---

51. *On the Action of Urari and of Strychnia on the Animal Economy.* By Prof. Albert Kölliker, of Würzburg. (Communicated to the Royal Society.)—The communication which I now offer to the Royal Society, contains a brief statement of the results of a series of experiments which I lately made on the action of the urari poison and of strychnia on the animal economy.

I. *Urari.*—The urari is the well-known poison from Guiana, also called Curare and Woorara. That which I employed in my experiments I owe to the liberality of my friend, Professor Christison, of Edinburgh. The following are the conclusions at which I arrived respecting its operation:—

1. The urari causes death very rapidly when injected into the blood or inserted into a wound; when introduced by way of the mucous membrane of the

intestinal canal, its effects are slow and require a large dose for their production, especially in mammalia. When applied to the skin of frogs it is altogether inoperative.

2. Frogs poisoned with very small doses of urari may gradually recover, even after it has produced complete paralysis of the nerves. Mammalia may also be restored, even after large doses, provided respiration is maintained artificially.

3. The urari, acting through the blood, destroys the excitability of the motor nerves. In frogs under its operation the terminal branches of these nerves within the muscles lose their excitability in a few minutes, whilst their trunks become affected an hour or two later. If, after the nervous extremities have become paralyzed, the heart of the animal be excised so as to prevent the nerves from receiving any further share of the poison, the nervous trunks may retain their excitability for three or four hours.

4. The brain is less affected by the urari than the nerves in the muscles; still when, by ligature of the two aortic arches, in frogs, the poisoning is confined to the anterior half of the body, the voluntary movements of the limbs speedily cease, while automatic movements, of doubtful nature and probably proceeding from the medulla oblongata, may be still observed for half an hour or an hour after the poison has begun to operate.

5. The spinal cord is considerably less affected than the brain by this poison, and by local limitation of the poisoning (as in No. 4) it is found that the cord retains its reflex activity from half an hour to an hour and a half, and the excitability of its white substance or its conducting power from two to three hours after the poison has taken effect. It is worthy of remark that in such cases the impaired reflex activity of the spinal cord may be revived by strychnia directly applied to it.

6. The sensory nerves, as shown also by locally limited poisoning, retain their functional activity as long at any rate as reflex actions can be excited, and when the depressed reflex activity has been revived by means of strychnia, these nerves are found not to have been in the slightest degree injured, so that it seems doubtful whether the urari in any way affects them.

7. The nerves of the involuntary muscles and of the glands are also paralyzed by the action of urari, at least I find this to be true in the following cases, viz:—

a. The pneumogastric, as regards its influence on the heart.
b. The sympathetic (its cervical portion), in its relation to the iris.
c. The nerves of the posterior lymph-hearts of the frog.
d. The nerves of the vessels in the web of the frog's foot.
e. The splanchnic nerves of the rabbit, as affecting the peristaltic motions.
f. The nerves governing the secretion of the submaxillary gland in dogs.

8. The voluntary muscles remain perfectly excitable, but show a greater tendency than usual to merely local contractions. In general the cadaveric rigidity of these muscles appears to set in later than usual.

9. The plain or non-striated muscles also remain long irritable after poisoning by urari.

10. The heart, in amphibia, is little affected by urari. Its pulsation as well as the circulation of the blood goes on regularly for many hours after the poisoning is established. The only thing worthy of note is that the beat of the heart appears to be somewhat quickened, probably from paralysis of the pneumogastric nerves. In frogs poisoned with urari, the heart, when cut in two, shows the usual phenomenon, namely, that the half which contains the ganglia continues to pulsate while the other does not; from which, it may be inferred, that these ganglia are not paralyzed. As to the nerves in the substance of the heart, those at least which are derived from the pneumogastric, are unquestionably paralyzed (vide No. 7).

11. The lymph-hearts of frogs poisoned with urari soon cease to move.

12. The blood of animals poisoned by urari is fluid and dark, but coagulates when drawn from the vessels, and forms a weak clot which is but little reddened by exposure to air. Directly mixed with blood, urari does not prevent

coagulation, but the blood in this case also remains dark, and scarcely reddens on exposure.

13. The blood of animals poisoned by urari has the same poisonous qualities as that substance itself, but not in a degree sufficient to produce the full effects of the poison. Urari when directly mixed with blood loses none of its efficacy.

14. Urari, in concentrated solution, applied locally to nerves extinguishes their excitability, but only after a considerable time, and it appears to act similarly on the nerves in the substance of the muscles. Dilute solutions have no injurious operation. Applied directly to the brain and spinal cord, urari is altogether harmless provided its absorption be prevented.

15. When artificial respiration is kept up in quadrupeds poisoned with urari, I find that, as observed by Bernard, many of the secretions become increased—as the tears, saliva, urine, and mucus of the air-passages, which effect appears to be owing to the paralysis of the vascular nerves and consequent dilatation of the vessels caused by the poison.

16. In mammalia urari causes death by paralysis of the respiratory nerves and suppression of the respiration, which brings on convulsions in these animals as a collateral effect. In frogs the final extinction of the functions may also be partly ascribed to suppressed action of the lungs and defective oxidation of the blood, which at length renders the heart unfit to perform its office; but it must be observed that in this case the cause of death is not so plain, inasmuch as in these animals the functions are in a great degree independent of the pulmonary respiration.

II. *Strychnia.*—Some experiments with strychnia (the acetate) gave the following results:—

1. Strychnia has not the least influence on the peripheral nerves through the blood, which is best shown by cutting the nerves before administering the poison.

2. Strychnia paralyzes the motor nerves of the voluntary muscles by exciting them to too energetic action, a paralysis which may be compared to that caused by powerful electric currents acting upon the nerves. In frogs, when the tetanic spasms are over, the nerves often show no trace of excitability; in mammalia they generally retain it in a slight degree, but never show the same energy of action as when uninjured.

3. Strychnia does not affect the sensory nerves.

4. The heart is not affected by strychnia, not even during the tetanic spasms, with the exception only that its pulsations are sometimes a little slower during the tetanic state. On the contrary, the lymph-hearts of frogs contract themselves as soon as the tetanus begins, and remain in this state as long as the spasms last.

5. The tetanic fits can be brought on in two ways: first, through the sensory nerves, which, by irritating the gray substance of the spinal cord, produce the tetanic contractions as reflex movements; and, secondly, through the brain, which is not affected at all by strychnia and preserves its powers of volition and sensation. Accordingly, animals poisoned with strychnia try to move in the ordinary way, but every attempt brings on a tetanic fit, so that it is plain that the spinal cord may also be excited by the brain to its peculiar actions.

6. If the tetanus produced by strychnia has been strong, the muscles are less irritable and pass much sooner into the state of cadaveric rigidity, which is very strongly marked, and seems to last longer than it generally does. The same early onset of rigidity may be observed in animals killed by tetanus excited by electricity.—*Med. Times and Gaz.*, Sept. 13, 1856.

52. *On Oils as Promoting the Poisonous Action of Cantharides.*—The solubility of cantharidin in oils has led Orfila, Taylor, Christison, Mitscherlich, Oesterlen, and other toxicologists to promulgate cautions against the use of fatty matters in poisoning with Spanish flies. On the other hand, Clarus, in his *Handbuch der Specielle Arzneimittellehre*, considers that there are no grounds for this caution. Professor Schoff, of Vienna, who has performed a number of experiments on the action of Spanish flies and cantharides, has published